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THE CANADIAN MEDICAL ASSOCIATION

Managing Editor: T. C. ROUTLEY, M.D., F.R.C.P.[C.]

Assistant Editors: M. R. DUFRESNE, M.D.

GORDON T. DICKINSON, M.D.

Editorial Offices: 150 ST. GEORGE ST., TORONTO

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ETIOLOGY OF PSYCHOSOMATIC DISEASE

Suburban living is becoming the accepted form for ever greater numbers of city dwellers. In the United States 12.4 million people were added to the suburbs from 1950 to 1958.¹ The rate of increase of the suburbs was three times the rate within the cities to which they are adjacent and more than double that of the United States as a whole. This is even more striking when we learn that some of the larger cities of the United States have in the same period grown only very little or not at all. Boston has had a decrease of population (1% per annum) and so have Pittsburgh and Providence, whilst New York City, Newark and Jersey City have shown no appreciable change. Their adjacent communities have at the same time been growing at the rate of 3.9% a year. Because of this suburban growth, the population of the metropolitan areas in the U.S.A. rose in 1958 to 59% of the total population of the country (56% in 1950).

It is obvious that such rapid growth of communities is bound to affect inhabitants in various ways, and sociologists have been warning us of its hazards. Gordon and Gordon² report a study on the prevalence of psychosomatic diseases in rapidly growing suburbs. Hospital admissions for asthma, coronary thrombosis, duodenal ulcer, essential hypertension and hypertensive cardiovascular disease were compared in three hospitals with the admissions for bronchopneumonia in the period of 1950-1951 and 1957-1958. The three hospitals were located in a rapidly growing suburb, a mixed rural community, and a stable rural area respectively. The results of this statistical study show a significant difference in the incidence of various diseases in the three hospitals. There was a higher percentage of admission of tension disorders to Englewood Hospital, serving a rapidly growing suburb, than to the other two. Asthma was more prevalent in the hospital serving a rural community with a high percentage of old people. "They are being

left behind there, are without adequate activities and are often quite depressed."

We are all aware of the present popularity of "psychosomatics" in both medical and lay circles, and it is not as difficult as it used to be to convince a patient that his disease is primarily in his "mind" and not in his "body". But that does not prove that the illnesses which are ascribed to emotional disorders are psychosomatic diseases. Let us remember that some 130 years ago general paralysis of the insane was regarded as the psychosomatic illness *par excellence*, the exact opposite of the present-day concept.

In a lecture to the Zurich Medical Association Bleuler³ stresses the difficulties of proving the purely emotional etiology of the so-called psychosomatic diseases. According to him, the well-known effects of emotion on bodily function do not prove that organic disease is actually caused by emotional disorder. Bleuler is a psychiatrist, who had initially been a general practitioner and a surgeon, and his observations are obviously based on personal experience. He welcomes continued investigation of the mechanisms by which emotional disorders can produce physical illness, but he points out that when one states definitely and accepts as proven that the above diseases are caused by emotional disturbance, one has left solid scientific ground and is in the realm of speculation. Somatic manifestations of psychiatric diseases must be sharply separated from the conditions under discussion and cannot serve as proof by analogy. The fact that we have no evidence of impersonal, extra-corporeal etiological agents of these diseases does not exclude our finding such at a later date. Human beings explain even impersonal events in a personal way, and it is not surprising that bodily illness is blamed on a mental distress. Uncovering of connections between inner experience and manifestations of illness makes assumption of psychosomatic disease plausible and possible, but does in itself not prove it. Psychotherapeutic success has indeed been achieved temporarily in many cases, but prolonged cures have been few and not sufficiently well documented by objective, sound medical observation. Bleuler sees danger in the vagueness of the concept of psychosomatics. It allows us to imagine the connections between psyche and somatic functions in any way we like and to have many misconceptions that are harmful to progress.

Although it has to be admitted that the present trend towards acceptance of psychosomatic medicine has promoted a more comprehensive approach to the patient and his illness, the empirically minded physician cannot rid himself of a sense of discomfort. The discussion by Gordon and Gordon of the reasons for the differences in the three communities sounds logical and their explanations are ingenious. Maybe the unaccustomed language of psychosomatics is incomprehensible to some of us. Or is it the lack of a sound, philosophical

basis? Perhaps the methods applied in this field are so radically different from those used by natural sciences. One may question whether the rate of admission to hospitals can be considered a true yardstick for the incidence of disease. As neither asthma nor peptic ulcer are diseases for which most patients are admitted to hospital, one could argue that an increased rate of admission merely indicates a more severe form of the diseases.

Will such studies be able to take all possible factors into consideration? Will they establish the etiological relationship between "modern high-pressure living" with its resulting emotional strains and stresses, and gastric hypersecretion, hypermotility and ulcer, or hypercholesterolaemia and coronary artery disease? Is it really as simple as that?

W. GROBIN

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Editorial Comments

GRISEOFULVIN: AN APPRAISAL

The deluge of papers¹⁻⁹ on griseofulvin makes some critical evaluation and condensation seem mandatory to establish a rational basis for the use of this new antifungal antibiotic in superficial fungous infections.

Keratin is made up of closely packed polypeptide chains held together by disulfide bonds of cystine. It is resistant to solvents and enzymes. The nails, hair cortex and cuticle are made of hard keratin; the stratum corneum of the epidermis and hair medulla are made of soft keratin. Superficial fungous infections are a parasitic infection of keratin; the fungi live in, feed on, distort and destroy keratin. Some fungi cause an acute inflammatory reaction which is self-destructive: the inflammation casts off or destroys the fungi or keratin. It should be noted that these are self-healing conditions. Those fungi which cause only a mild reaction frequently give rise to a chronic low-grade infection. The basic reason for individual susceptibility is not known. Some of the known conditioning factors are heat, moisture (tinea pedis), obesity, diabetes mellitus (moniliasis) and Cushing's syndrome¹⁰ (*Trichophyton rubrum* infections). The basis for treatment prior to griseofulvin was a peeling off of invaded keratin by ointments (Whitfield's ointment) or x-ray epilation. This was curative in some cases and helpful in others. However, in many cases the anatomy of the skin appendages (e.g., hair and sides of nails) made thorough peeling impossible, and so the invaded keratin was never completely removed.

Griseofulvin was isolated in 1939 from the mould *Penicillium griseofulvin*. It was used successfully in

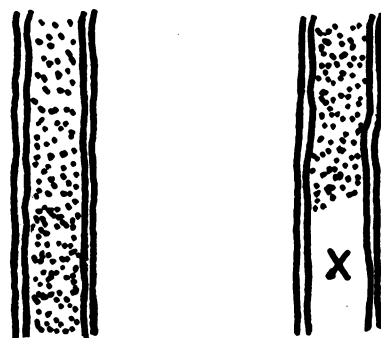


Fig. 1.—Diagram of cross-section of human hairs infected by *Microsporion canis*. X marks the proximal portion of the hair where the griseofulvin-containing keratin has no fungal products. (After A. R. Birt *et al.*⁶)

the treatment of fungous diseases of plants and guinea pigs before Williams, Marten and Sarkany² reported its successful use orally in human ringworm. The drug is absorbed from the gastro-intestinal tract into the blood stream, from which it is deposited into the growing keratin of the skin and hair. Fungi do not grow in griseofulvin-containing keratin (see Fig. 1). We can postulate the development of a griseofulvin shield which slowly extends into the non-living keratin of the nails and hair. This antibiotic is fungistatic, and the rate of cure will depend on the rate of growth.

Keratin source

Time for response

Skin	1 week
Hair	5-6 weeks
Nails: Finger	5 months
Toe	9 months or more

Griseofulvin must be given continuously because re-infection may occur from the tips of infected hairs not infiltrated with the drug or from the keratotic subungual debris in the nails.

Many problems about the practical therapeutics of this drug are not yet understood. The following material is based on a thorough study of what has so far come to light. As with all medical practice, an accurate diagnosis is essential. Clinically, even the most expert student of morphology can be led up the garden path unless there is confirmatory laboratory evidence of fungous disease. This evidence may be obtained by examination under the microscope of skin scrapings or hair soaked in 15% KOH, by culture on Sabouraud's medium or by examination under an ultra-violet (Wood's) light. Most provincial health departments provide these services free of charge. Therapeutic trial of this powerful specific drug are to be condemned. A superficial fungous disease is not an emergency. The daily oral dose in children up to 12 is 25 mg./kg., in adults 1.0 g. The drug is not effective topically. Griseofulvin is the drug of choice in those superficial fungous diseases most resistant to peeling agents and those least influenced by conditioning factors. *Trichophyton rubrum* is a chronic low-grade infection of the nails and skin. Prior to griseofulvin there was no effective treatment. *Microsporion audouini* (human non-inflammatory) scalp ringworm was satisfactorily treated only by x-ray epilation,